increased ejection fraction failed to show a significant difference ($p = 0.2045$).

Patients with congestive heart failure and a baseline ejection fraction ≤35.5% have been reported to show a lower survival rate than patients with an ejection fraction >35.5% (3), and an increase in left ventricular ejection fraction after treatment with a vasodilator may be an important marker for improved survival (4). Because it has been reported that one long-acting ACE inhibitor (enalapril) has shown an improved survival rate in patients with severe congestive heart failure (5), a retrospective look at the subgroup of patients with a baseline ejection fraction ≤35% was identified as an interesting analysis.

In the subgroup of patients with a baseline ejection fraction ≤35%, the mean change from baseline in the ejection fraction for patients in the long-acting ACE inhibitor group should have been 4.4% (not 4.1%) and that for patients in the short-acting group should have been 0.6% (not 0.8%). The mean change from baseline in the ejection fraction was significant ($p < 0.0001$) for the long-acting ACE inhibitor group and not significant for the short-acting group. The long-acting agent was significantly ($p = 0.0153$; Student's t test) better than the short-acting one in increasing the ejection fraction of patients with congestive heart failure and a baseline ejection fraction ≤35%.

Functional capacity and quality of life evaluations (Yale scale dyspnea/fatigue index [6]) showed improvement from baseline for both short- and long-acting ACE inhibitors; however, the long-acting agent was superior ($p < 0.05$) to the short-acting one. All signs and symptoms of congestive heart failure were significantly improved with the short- and long-term agents, with the exception of rales and jugular venous distension, which were not improved from baseline for the group taking the short-acting agent.

Our paper concludes that both short-acting and long-acting ACE inhibitors have beneficial effects for patients with congestive heart failure concomitantly receiving diuretics or digitalis, or both. The data also show that a long-acting ACE inhibitor is more effective than a short-acting one, as shown by increased treadmill exercise duration (particularly in patients with renal impairment or the elderly), increased ejection fractions (particularly in those with a baseline ejection fraction ≤35%) and improved functional capacity. I agree that more comparative trials are required to further delineate the differences among the various ACE inhibitors in the management of patients with congestive heart failure.

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Postinfarction Risk Stratification

We found the excellent article by Krone et al. (1) of particular interest because it confirmed the findings of our subsequent article in the Journal (2). We both found that exercise-induced ST segment depression predicted an increased risk in patients without Q waves on their electrocardiogram (ECG) at the time of exercise testing after myocardial infarction as opposed to a mixed sample including patients with Q waves (3). Our two articles explain why previous follow-up studies have not yielded consistent results regarding the risk of exercise-induced ST depression (4); differences could be due to the mix of patients regarding their ECG patterns at rest.

Krone et al. (1) found, however, that the risk of exercise-induced ST depression was only present in patients with pulmonary congestion during admission. Twenty-six (23%) of their 111 patients had pulmonary congestion by chest radiograph or physical examination, and exercise-induced ST depression generated a risk ratio of 6 for death in this group. Fortunately, these data were available on our patients, though not presented in our paper. Fifteen (27%) of our 55 patients without Q waves had pulmonary congestion using the same criteria as Krone et al. (1). Only 2 of the 11 deaths occurred in patients with pulmonary congestion (both also had exercise-induced ST depression). Thus, the risk ratio of 11 for exercise-induced ST depression in this group was not isolated to those with pulmonary congestion. Other events including interventions or noncardiac deaths did not explain this difference.

Two inconsistencies in their article are the following: Table 4 should read exercise features in "26 patients" rather than "85"; and the last sentence in the top paragraph on the right side of page 34 should read "without ST depression" rather than "without pulmonary congestion."

Differences between our papers include the following: in their study, 111 patients underwent an exercise test, 4 died and 8 had reinfarction during 1 year follow-up. Perhaps our findings will agree when they complete a second year of follow-up and accrue more end points.

The study of Krone et al. (1) and our study agree that exercise-induced ST depression generates a high risk in postinfarction patients without Q waves, but are in disagreement as to the effect of the presence of pulmonary congestion. Our conclusion is that the rest ECG has a profound effect on the risk posed by exercise-induced ST depression in patients tested after myocardial infarction; that is, the exercise ECG is excellent for identifying a high risk group in patients without Q waves, but probably does not represent ischemia (5) and is not associated with increased risk in other Q wave patterns.

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Reply

We thank Froelicher for the opportunity for this dialogue (we apologize for the two typographical errors).

Our major finding was that by using clinical risk stratification—namely, the presence or absence of pulmonary congestion—we could identify a group with a mortality risk so low that early low level exercise testing was of no value in further stratifying patients. Other studies (1–4) have found pulmonary congestion to be an important determinant of risk. Why this does not appear to be the case in the Veterans Administration population is not clear; that population [Ref 2 of Froelicher] appears to be unique, with an unusually high postinfarction mortality rate. Our 2 year mortality rate in the patients with non-Q wave infarction who exercised was 5%. It may be that other factors, not measured, such as adherence to a healthy life-style after discharge or cessation of smoking (5), distinguish the Veterans Administration patient population from the population of other studies.

In our high risk group, with a mortality comparable with the overall mortality in the Veterans Administration group, ST depression with exercise was an important prognostic factor. Our data suggest, however, that even those without ST depression in this high risk group had a high incidence of cardiac death or cardiac events in the first year.

The fact that our studies differ in some details is not surprising and serves to demonstrate the strengths and weaknesses of studies of this type. Both studies are retrospective, post hoc reviews of data, and the precise factors identified (ability to exercise and pulmonary congestion) were identified retrospectively as important stratifying variables. Thus the results of each study can be considered only as "hypothesis generating," not definitive. In addition, we have demonstrated that, although some statements developed in this way can be generalized to other populations, the populations may be different in some unknown way. The question the clinician dealing with the individual patient must answer is whether a negative test identifies a patient at a low enough risk that conservative therapy can be utilized. Our data suggest that, although ST depression with exercise in a high risk group identifies a subset at especially high risk, a negative exercise test in a high risk group cannot identify a patient at low enough risk that further evaluation is not required.

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